

SHORT REPORTS

Physiological response to parachute jumping

The metabolic and hormonal responses to stress in racing drivers, airline pilots, rock climbers, public speakers, and parachutists have been reported.¹ Most studies have concentrated on catecholamine concentrations and their effects on heart rate; little information is available about changes in the concentrations of other hormones and intermediary metabolites.

Subjects, methods, and results

Eight healthy subjects (two women, six men) took part in the study. None had made a parachute jump before. Their ages ranged from 23 to 29, and all were within 10% of their ideal body weight. All subjects fasted from 1300 and jumped between 1900 and 2100. Venous blood was taken through an indwelling cannula before they left for the airfield (basal values); on arrival; after 30 minutes' exercise training on the ground; in the aircraft at 610 m immediately before they jumped from a static line; within 30 seconds after landing; and at 10, 20, and 30 minutes after landing. A Tracker Holter monitor was attached to each subject to give a continuous electrocardiogram, and the tapes were analysed on a Pathfinder 3 analyser.

Concentrations of intermediary metabolites were measured fluorimetrically and spectrophotometrically (acetoacetate). Non-esterified fatty acid concentrations were measured by a radiocobalt soap method, potassium concentrations by flame photometry, and hormone concentrations by radioimmunoassay or high performance liquid chromatography (catecholamines).² Data were classified by subject and time and analysed by two way analysis of variance. When the variance ratio for a time effect was significant the change from basal values was assessed by Student's *t* test for paired samples.

All eight subjects showed three clear peaks in heart rate: on arriving at the airfield, during ground training, and on jumping out of the aeroplane (the greatest increase). Heart rates increased from a basal mean of 78 (range 62-91) beats/min to a peak of 160 (150-183). No ventricular arrhythmias or ST segment abnormalities were recorded.

The table shows the changes in concentrations of intermediary metabolites and hormones. In six subjects peak concentrations of non-esterified fatty acids were greater than 1.4 mmol/l, and three had lactate concentrations greater than 5 mmol/l. Concentrations of adrenaline and noradrenaline rose in parallel, reaching peak values when the subjects were in the aeroplane and on landing, respectively. After landing the concentration of adrenaline fell to basal values within 10 minutes but the concentration of noradrenaline was still significantly raised 30 minutes later ($p < 0.05$).

Comment

The metabolic response to the stress of parachute jumping seems similar to the response to other forms of stress studied. The rise in concentrations of glucose and fatty acids with a fall in the insulin concentration results from increased concentrations of circulating catecholamines. We observed a rise of three to 15 times the basal value in both adrenaline and noradrenaline concentrations in the aircraft immediately before the jump.

The popularity of parachuting is increasing, especially now that people are often sponsored for jumping. In our fit young subjects the heart rate rose to a mean of 160 beats/min; concentrations of catecholamines increased fivefold and those of non-esterified fatty acids reached values associated with cardiac arrhythmias.³ In subjects with undiagnosed coronary artery disease the

demands on the heart of a sustained tachycardia, a low grade hypoxia caused by reduced oxygen pressure depending on the height at which the jump is made, and a simultaneous rise in the concentrations of catecholamines and non-esterified fatty acids could cause cardiac arrhythmias.⁴ Doctors asked to perform medical examinations to assess fitness to jump should be aware of the extreme nature of the stress of parachute jumping.

We are grateful to Mr M Bonney and Reynolds Medical Ltd, Hertford, for the loan of the electrocardiographic monitors and for the analysis of tapes, and to the bicentenary advisory board of the General Hospital, Birmingham, for financial help.

- 1 Carruthers M. Field studies: emotion and beta blockade. In: Christie MJ, Mellett PG, eds. *Foundations of psychosomatics*. Chichester: John Wiley, 1981:223-39.
- 2 Garden G, Hale PJ, Horrocks PM, Crase J, Hammond V, Nattrass M. Metabolic and hormone responses during squash. *Eur J Appl Physiol* 1986;55:445-9.
- 3 Oliver MF, Kurien VA, Greenwood TW. Relation between serum free fatty acids and arrhythmias and death after acute myocardial infarction. *Lancet* 1968;ii:710-5.
- 4 Tansey MJB, Opie LH. Relation between plasma free fatty acids and arrhythmias within the first twelve hours of acute myocardial infarction. *Lancet* 1983;iii:419-21.

(Accepted 26 May 1987)

Diabetic Clinic, General Hospital, Birmingham B4 6NH

R ANFILOGOFF, BA, MB, house physician
P J HALE, DM, MRCP, senior registrar
M NATTRASS, PHD, FRCP, consultant physician

Department of Medicine, University of Newcastle upon Tyne

V A HAMMOND, BSC, PHD, research biochemist

Leamington Spa, Warwickshire

J C CARTER, MB, MRCP, general practitioner

Correspondence to: Dr Nattrass.

Endogenous digoxin-like immunoreactivity in congestive heart failure

Material cross reacting with antidigoxin antibodies has been found in several clinical settings, including the third trimester of pregnancy, the neonatal period, liver disease, and chronic renal failure.¹ Though the exact nature of this material is not known, we have shown recently that digoxin-like immunoreactive substance is probably a steroid.²

Several observations have suggested a pathophysiological role for digoxin-like immunoreactive substance. Firstly, it has been found in several hypertensive states, including toxemia of pregnancy and experimental hypertension in rats. Secondly, the hypertension in aortic coarctate rats is reversed by antidigoxin antibodies.³ Thirdly, gel filtration fractions of serum which contained digoxin-like immunoreactive substance from volume

Mean (SD) concentrations of intermediary metabolites and hormones in eight subjects before and after a parachute jump

	Basal	On arrival at airfield	After exercise training	In aeroplane immediately before jump	On landing	10 Minutes after landing	20 Minutes after landing	30 Minutes after landing
Glucose (mmol/l)	4.9 (0.3)	4.6 (0.4)	4.6 (0.5)	5.3 (0.3)	5.8 (0.5)**	6.2 (0.6)**	5.7 (0.4)**	5.3 (0.3)*
Lactate (mmol/l)	0.84 (0.12)	0.75 (0.17)	1.13 (0.51)	1.47 (0.36)	4.48 (1.39)**	3.32 (1.02)**	1.66 (0.70)*	1.08 (0.21)
Pyruvate (mmol/l)	0.07 (0.01)	0.06 (0.02)	0.09 (0.05)	0.09 (0.02)	0.18 (0.04)**	0.18 (0.05)**	0.10 (0.02)*	0.08 (0.01)
Alanine (mmol/l)	0.31 (0.05)	0.28 (0.03)	0.31 (0.05)	0.27 (0.04)	0.29 (0.05)	0.31 (0.05)	0.29 (0.04)	0.27 (0.04)
Non-esterified fatty acids (mmol/l)	0.60 (0.12)	0.75 (0.29)	0.93 (0.10)**	1.47 (0.37)**	1.45 (0.38)**	1.43 (0.28)**	1.06 (0.23)**	1.06 (0.25)**
Glycerol (mmol/l)	0.07 (0.03)	0.10 (0.06)	0.15 (0.05)**	0.15 (0.06)**	0.21 (0.09)**	0.23 (0.09)**	0.15 (0.05)**	0.13 (0.04)**
Total ketone bodies (mmol/l)	0.05 (0.03)	0.10 (0.06)	0.14 (0.05)	0.35 (0.24)**	0.34 (0.23)**	0.30 (0.20)**	0.25 (0.15)*	0.25 (0.17)*
Adrenaline (nmol/l)	0.50 (0.26)	0.57 (0.19)	0.82 (0.37)	2.21 (1.06)**	2.11 (1.11)**	0.71 (0.17)	0.56 (0.20)	0.41 (0.21)
Noradrenaline (nmol/l)	1.76 (0.58)	2.40 (0.43)	2.66 (0.55)	4.24 (1.23)**	6.35 (1.93)**	4.87 (1.38)**	4.46 (1.33)**	3.35 (0.90)*
Insulin (mU/l)	6.79 (3.81)	3.03 (1.55)*	2.34 (1.41)**	3.31 (0.74)*	4.35 (2.96)	7.65 (1.54)	5.91 (3.61)	4.11 (1.26)*
Growth hormone (mU/l)	3.06 (3.28)	1.39 (0.56)	17.00 (6.16)**	4.18 (4.08)	5.21 (6.47)	6.69 (5.90)	8.53 (5.63)*	6.30 (4.39)
Cortisol (nmol/l)	359 (120)	248 (141)	221 (197)	466 (289)	561 (333)*	680 (327)**	685 (334)**	632 (354)**
Prolactin (mU/l)	280 (129)	188 (93)	193 (114)	453 (328)*	648 (529)**	505 (271)**	356 (228)	296 (170)
Potassium (mmol/l)	3.9 (0.2)	3.8 (0.4)	3.7 (0.2)*	4.3 (0.4)**	3.6 (0.2)*	3.8 (0.2)	3.6 (0.3)*	3.8 (0.2)

Significance of change from basal value: * $p < 0.05$, ** $p < 0.001$.